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DISEASES OF THE CHEST

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C. M. HENDRICKS, EDITOR-IN-CHIEF

(A MONTHLY PUBLICATION)

"The most important factor in diagnosis in the majority of cases of pulmonary tuberculosis is keeping the disease in mind."
Lawrason Brown, M. D.

Editorial Comment

President's Message DISEASES OF THE CHEST comes to you this month as the first issue of a completed organization. For several years a number of physicians prominent in tuberculosis work have felt the need of a journal that will bring to the physician in general practice a wider conception of the principles underlying the early diagnosis of tuberculosis. It has been recognized by this group of men that if tuberculosis is to be eradicated it must be done thru the efforts of the family physician. They realize that he is the only physician who has the opportunity of seeing the incipient or moderately advanced case.

August 10th, these men met in Albuquerque, New Mexico, and at a most enthusiastic meeting formed a permanent organization and elected officers. This organization does not belong to any local group but is composed of men from widely scattered sections of the United States. The essential qualification specifies that each member in this group be a man interested in tuberculosis work but not affiliated with any federal, state, or municipal sanatorium. One of the objects of the federation is to bring before physicians the important part played in the treatment of tuberculosis by the private or semi-private sanatoria.

It is felt that DISEASES OF THE CHEST will not encroach on the work of any other journal, inasmuch as it will be sent to men who are in general practice and do not restrict themselves to the treatment of tuberculosis.

The papers will be written by physicians who have had many years experience both in treating cases and as writers on the subject. It is the desire to make these papers interesting to the general practitioner and to eliminate as far as possible subjects of ultra-scientific nature.

We hope you like DISEASES OF THE CHEST and will not hesitate to request papers on any phase of tuberculosis work in which you are especially interested.

WM. DEVITT, M. D.

Food Handlers PHYSICAL EXAMINATION of food handlers is required by law in practically every city of any size in the United States. Many states have similar laws. Such laws are the result of the diligent work of the medical profession along the lines of preventive medicine, but, strangely, the profession has been negligent and non-coop-

erative in the enforcement of the law. As a result the examination has fallen into the province of the public health authorities, and the income therefrom has been lost to the private doctor. In many cities the examination has actually been abandoned because of the laxity with which the profession has carried on this work.

Compensation for this examination has apparently not been enough to interest the conscientious physician, or the physician has taken the fee and filled in the necessary blank after a half-hearted examination. A glance at the pharynx and a casual glance at the external genital organs have apparently been considered an examination which would reveal contagious or infectious disease. Venereal diseases are not transmitted in food.

This is a plea for careful examination of every food handler. If the examiner does nothing else he should take the necessary few minutes to carefully examine the worker's chest. Tuberculosis is one disease which can be spread from one person to another in food, or on utensils at restaurants, or soda fountains, etc. Obviously the discovery of active disease is the discovery of a possible source of infection. How many such sources have been allowed to dispense food to the unsuspecting public behind a lax food-handler's certificate is a problem for a mathematician. That such conditions can be prevented must certainly be obvious even to the laity.

The medical profession today has been criticized from many angles. It deplores such criticism, and it deplores its present status—that of being on the very verge of a change in the old order of things. May we be so bold as to suggest that the profession is responsible to a great extent for its present circumstances? May we further suggest that we have failed in one respect in our attitude toward food-handlers certificates? Laws are made to be enforced, and this is one law that the doctors were called upon to enforce. Let us give each food handler a thorough examination and fulfill our duty as the guardian of the people's health. R.B.H., JR.

Educate the Patient

IN TUBERCULOSIS there are no secrets to be kept from the patient. Frankness upon the part of the physician may shock the patient slightly at first, but it will be much appreciated as time goes on.

Usually the more knowledge the patient has concerning his condition, the more clearly he understands the reasons for carrying out the details of treatment which have been prescribed for him, the better he will co-operate with his physician and the quicker he will secure an arrestment of his trouble.

Education of the patient, therefore, is perhaps the most important and should be one of the first steps in the treatment. This may be brought about in two ways:

1. Send the patient to a good sanatorium even if he cannot afford to stay more than a month. Here he will learn a lot as a result of the routine which he is required to carry out, from the instructions and information imparted by the physicians at the sanatorium, and from association with other and more experienced patients in the institution.

2. See that he is supplied with authoritative and reliable literature concerning tuberculosis. There is now a great abundance of such literature available. A splendid assortment of books and reprints may be obtained from the National Tuberculosis Association, New York. One of the best of them is the concise booklet bearing the title of "What You Should Know About Tuberculosis." Of the books

which go more into detail concerning the disease, a most excellent one has recently been written by Dr. Fred G. Holmes of Phoenix, Arizona. Another is by Dr. Lawrason Brown. There are numbers of others, all well worth reading, and any of them may be purchased through the National Association.

Other excellent reprints may be obtained from the Metropolitan Life Insurance Company of New York. This Company, by the way, should be held in the highest esteem by both the laity and the medical profession because of the excellent publicity which they have given in their advertisements to the facts concerning tuberculosis and other preventable diseases. Doubtless this publicity has had much to do with the reduction in the mortality rate of tuberculosis.

Another very commendable thing which the Metropolitan Life has done is the establishment of a large modern sanatorium for the care of its employees who develop tuberculosis.

This, by the way, puts its stamp of approval upon sanatorium treatment both as to the results obtained and to its economy in the end. R.B.H.

Heliotherapy THERE is a widespread belief among doctors, as well as the laity, that heliotherapy is not to be prescribed for patients with pulmonary tuberculosis, but that it is of great benefit to those who have the extrapulmonary form of the disease. In a way this tradition is fortunate, because it undoubtedly tends to prevent the tyro from doing an endless amount of harm. On the other hand, however, patients with pulmonary tuberculosis fail to receive the great benefit they may derive from heliotherapy, given at the proper time and in the proper dosage.

It is certainly well recognized that almost every patient with extrapulmonary tuberculosis has, in addition, a more or less marked pulmonary lesion. The question naturally arises, why is it that cases with multiple tuberculous lesions, including those of the lungs, will respond favorably to heliotherapy, while those with pulmonary lesions alone cannot safely take the treatment. The answer, of course, is that they can, provided the physician understands the limitations and dangers of heliotherapy.

The above mentioned tradition has probably grown up because of the serious consequences often following over-dosage of natural or artificial light treatments. An over-dose may produce a reaction of a lesion localized in some bone or joint, but on account of the anatomical structure of the tissues harboring the lesion this reaction subsides usually without serious consequence. On the other hand, a pulmonary lesion, which is activated by over-dosage of light, often has no firm barrier to prevent the progress and extension of the activation, and grave consequences ensue. Not infrequently when an extra-pulmonary lesion develops, the pulmonary lesion shows a marked tendency to recession, and this may, in part, explain the fact that patients with extra-pulmonary tuberculosis frequently develop pulmonary exacerbations under heliotherapy treatment.

Granted that the above reasoning is true, it would certainly seem justifiable to use heliotherapy in properly selected cases of pulmonary tuberculosis. Such treatment is advisable, however, only when the patient is under close observation and in the hands of one thoroughly experienced in the use of light therapy. Almost invariably the patient has the feeling that if a little light does good, more will do better, and unless he is very closely observed he is extremely apt to resort to over-dosage.

It can safely be said that most patients with pulmonary tuberculosis, if they reach a stage where the lesion is healing and where they are free from active symptoms of toxemia, can be given heliotherapy with marked benefit. A.M.F.

An Operation For Tuberculous Empyema*

BY
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THE ORDINARY form of rib resection with introduction of a drainage tube is notoriously unsatisfactory in tuberculous empyema, especially in empyemas the pus of which contains either tubercle bacilli alone or no demonstrable organism at all; i.e., empyemas not secondarily infected. So called "closed" drainage, drainage by means of a tube introduced into an intercostal space through a trocar, is equally bad. Both of these methods always lead to secondary infection and almost always to fever, sepsis, and death.

Some forms of tuberculous empyema, however, occasionally purely tuberculous ones, more often secondarily infected ones, produce such consistently high swinging temperatures and such severe symptoms of toxicity, that surgeons are impelled again and again to drain in spite of previous warnings and in spite of full knowledge of the trouble that usually ensues.

Several methods of treatment are proposed for such toxic tuberculous empyemas. Aspiration with a syringe, replacement of the pus by an equal quantity of air, and irrigation of the pyopneumothorax cavity with countless varieties of antiseptic solutions have been recommended. Aspiration often results in abatement of fever and sepsis for a few days and even weeks, but usually needs be repeated so often and so long that both the doctor's and the sufferer's patience is exhausted. At times it leads to cure, usually not.

Oleothorax (injection of gomenol and various other antiseptic or aseptic oils) has led to good results in some hands (Matson); to disastrous results in others.

Extensive thoracoplasty may succeed in obliterating the empyema cavity, but the operation needs be extensive and formidable. It entails considerable risk to patients weakened by long toxicity and considerable crippling at best.

Cure of these tuberculous empyemas is made difficult by the tenacity with which the underlying lung resists expansion. It is also made difficult by the unfavorable effect of an inlying drainage tube of any kind. The presence of the tube itself, I think, tends to keep up fever and sepsis. Some tuberculous empyemas treated ill advisedly by rib resection and drainage heal if one does nothing more than remove the drainage tube.

A desire to obviate a drainage tube led to an operation which has proved of use in a number of secondarily infected tuberculous empyemas. Encouraged by its success it has with some hesitancy recently been used in a few obstinately toxic tuberculous empyemas, in which no pus forming organisms could be found.

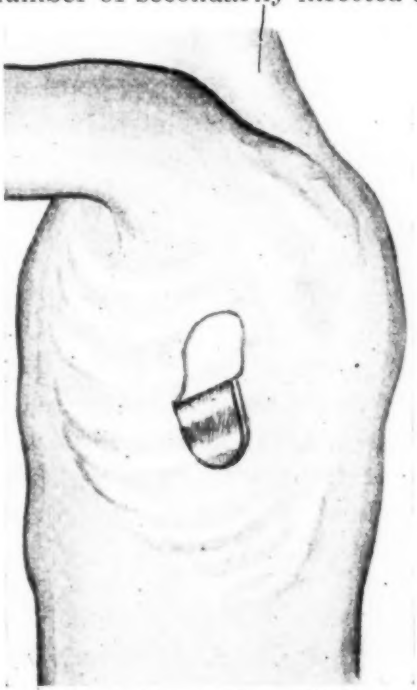

Fig. 1. Flap of skin outlined.

The operation causes the underlying lung to expand; it is, therefore, *not at*

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†From the Stanford Surgical Service of the San Francisco Hospital, Department of Public Health.

plicable to those empyemas in which the lung is so badly affected that expansion to any degree seems inadvisable. The question of applicability must be decided

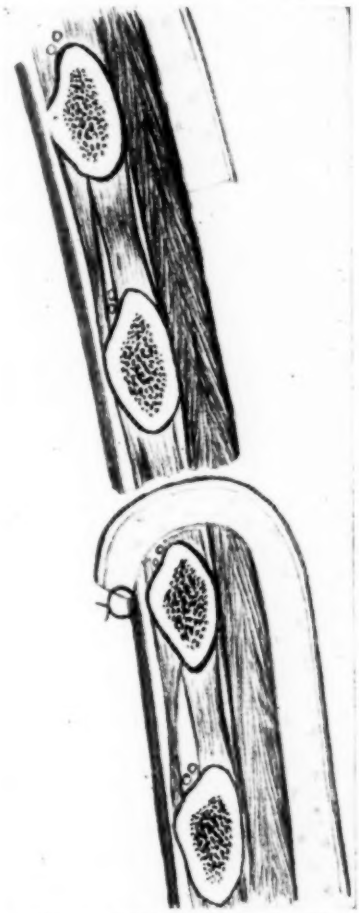

Fig. 2. Cross section of chest wall showing skin flap turned into chest and attached to pleura.

by clinical pulmonary symptoms and especially by X-rays taken prior to the appearance of the complicating empyema. If cough and expectoration of bacillus bearing sputum still persist and if early roentgenograms reveal large cavities or other extensive parenchymal damage, some form of thoracoplasty is probably safer. Fortunately, in many empyemas the lung seems little if at all affected and partial if not total expansion seems allowable. If but partial expansion seems advisable the operation to be described should be complemented by an upper thoracoplasty.

Technique

Under local anæsthesia a U-shaped flap of skin and subcutis is outlined about half way between the posterior axillary line and the line of the inferior scapular angle. The flap has a base about 2 inches wide which lies about one rib higher than the bottom of the empyema cavity, so that the rising diaphragm may not stop drainage; it is about 2½ inches long, the length of two ribs and their intercostal spaces, long enough to reach into the pleural cavity without the least tension and longer, therefore, in fat patients than in lean ones. The rib underlying the top of the flap is resected, the amount resected equalling the width of the flap. If the flap is too narrow and the resection too scant, drainage will be insufficient. It is good, I think, to strip the rib with a cautery instead of a raspatory and to inject the bared intercostal nerve with 1 cubic centimeter of absolute alcohol. The tip of this flap is turned into the chest and tacked to the pleura with one or two chromic catgut stitches; the edges of the defect in the skin are approximated with a few stitches of silkworm.

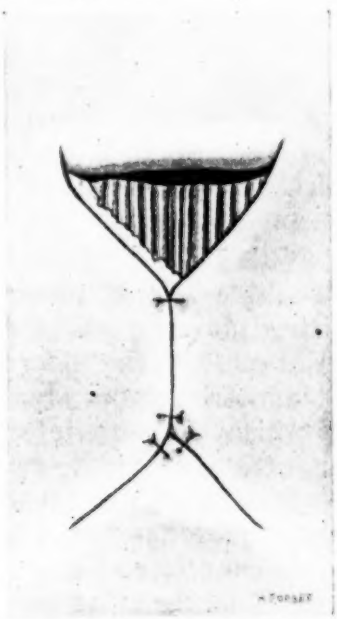

Fig. 3. Skin flap turned into chest cavity; edges of defect approximated by sutures.

This thoracotomy needs no tube, for the skin flap which lies against the soft parts of the chest keeps the wound open. It remains open until the lung reaches the chest wall, after which it spontaneously and automatically closes without further ado. It has a valve action; each cough or rise in intrapulmonary air pressure expels

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The Cause of Tuberculosis

BY
A. R. MASTEN, M. D.
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SINCE REMOTE ANTIQUITY tuberculosis has been taking its annual toll of human life. Many of the Egyptian mummies of 3,000 years ago show signs of bone tuberculosis. Since that early time this dread disease has received many names and has been laid to many causes. The Greeks called it "phthisis," meaning "wasting." Tuberculosis of the lungs has long been known as "consumption." Both of these names are entirely proper. Tuberculosis in all its forms is frequently spoken of as "the great white plague" or the "captain of the men of death." Since ancient times a few physicians have believed tuberculosis to be a contagious disease. The accuracy of this contention was verified in 1864 when J. A. Villemin demonstrated experimentally that the disease can be transmitted from one animal to another, and from human beings to animals by inoculation of the sputum brought up by tuberculous patients. It was not till 1882, however, that Robert Koch discovered the real cause of tuberculosis and proved it to be a specific kind of bacteria, the tubercle bacillus.

There are three types of tubercle bacilli which produce disease: (1) the human, which causes most of the tuberculosis in man; (2) the bovine type, which attacks cattle but which can also infect human beings; and (3) the avian, which is the usual type in birds. Tuberculosis of the bovine type of germ has now largely been eliminated by close inspection and testing of milch cows; and the avian type of germ seldom, if ever, attacks man. It is, therefore, the human type of tubercle bacilli which produces practically all cases of tuberculosis in man. Tubercle bacilli are very small, rod-shaped organisms which die if exposed for several hours to sunlight and fresh air. They are very resistant to drying and freezing, however, and can live a long time in poorly lighted and poorly ventilated surroundings. Tubercle bacilli enter the body on minute particles of dust, or on the tiny driplets of sputum emitted during a cough. It is from the sputum of some person with active tuberculosis that other persons become infected. It has been said, and rightly so, that if all persons sick with tuberculosis could be so carefully isolated that none of their germ-containing sputum would be carried to other people the disease would be eradicated in one generation. That is to say, every case of tuberculosis comes from another case of tuberculosis, it never originates spontaneously, and no other disease ever turns into it.

Tuberculosis was formerly thought to be hereditary, that is, the child of tuberculous parents was born with the disease or at least was born with a tendency to develop tuberculosis, a tuberculous diathesis, as it was called. This idea has now been proved to be a fallacy, for on thorough study it has been shown that children are practically never born with the disease, but instead acquire it after birth by receiving tubercle bacilli from their parents. Instead of inheriting a tuberculous diathesis, many investigators now believe quite the contrary, namely, that children of tuberculous parents inherit more resistance to the disease than other children. The reason tuberculosis runs in certain families is that in close family association it is easy to spread the germs from one member to another.

In civilized communities the contact between individuals is so close that sooner or later practically every person becomes infected with tuberculosis. This does not mean that every one acquires the disease, but only that some tubercle bacilli gain a foothold in their bodies. This first infection with tubercle bacilli is usually overcome without the person's knowing anything about it. Active disease occurs later when the person becomes debilitated, run-down, or else acquires a new infection with tubercle bacilli. This secondary in-

fection is the one that produces active tuberculosis, the disease known as consumption. It is this reinfection type of disease which produces so much disability and death. In the United States the incidence of tuberculous infection has diminished markedly in the last few years. In a study of children in Minnesota, Myers found a decrease of almost fifty percent between the years 1921 and 1933. Myers maintains that in many parts of the United States 90% of the boys and girls reach the teen age without being infected with tubercle bacilli. This is a vast improvement from the former condition when it was said that 95% of all children became infected before reaching thirteen. Myers also proved that of the children who react positively to tuberculin some ten percent will fall ill from clinical tuberculosis by the time they reach the age of 21 years; while only some 2% of the non-reactors will become ill with clinical tuberculosis. Formerly, it was believed that all tuberculosis was contracted in early childhood, but recent investigations, such as the one mentioned above in Minnesota, have shown that tuberculosis can be acquired at any age, and that both children and adults react in the same way to a first infection.

There are several possibilities inherent in the first infection which determine its eventual effect on the individual: (1) A person may receive a very small number of germs from a chance contact with someone having active tuberculosis, and thereafter receive no more germs for a long period of time. In this instance, the germs invariably produce a very slight disease process which tends to resolve and calcify (heal permanently). In this case the individual will probably go through life without any further trouble. In other words, a slight infection will produce enough immunity to protect the individual from the usual infections received during everyday life. This is what happens to the great majority of people. (2) A person may receive a somewhat larger number of germs at rather infrequent intervals, as might occur when some member of his family has a chronic case of tuberculosis and discharges germs only when he has a cold or otherwise becomes run-down. In this instance the disease process is usually more extensive than in the first class and it heals only partially, leaving a constant focus of disease. This focus may later undergo reactivation, because of a breaking down of the healing process, caused by lowered vitality of the individual. In this case the person becomes sick, because of a reactivation and spread of the germs which are already in his body. Most cases of active tuberculosis are thought to be of this type. Even in this type of infection active disease does not always result, for in perhaps the vast majority of people occurrences do not transpire which result in a marked lowering of their vitality, and they go through life without ever acquiring active consumption. (3) A person may receive a great number of germs at one time, a massive infection. In this instance the first infection is often severe and may progress steadily into the reinfection type of disease. This same result is obtained when a person receives small infections continuously, as when he is in close contact with someone having active tuberculosis who does not take great care to prevent his germ-containing sputum being transferred to all his associates. (4) A person may receive a small infection which heals completely as in class (1) and at some later date receive a massive or overwhelming reinfection from some outside source. In such a case the immunity obtained from the first infection is overcome and the individual develops active tuberculosis. Some investigators believe that this is the way most active cases of tuberculosis develop. They maintain that the primary infection completely heals in the vast majority of cases and that active consumption is the result of a reinfection from some exogenous (outside) source. That is to say, consumption is not generally the result of a lighting up of an old infection received in childhood.

The extent of the tuberculous process which results from any given infection is not controlled solely by the number of

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Gastro-Enterology and Pulmonary Tuberculosis

BY
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IT IS NOT the purpose of this discussion to set forth the more technical phases of the gastro-enterological picture of pulmonary tuberculosis which can be found by reference to the proper articles, but rather to present the more pertinent clinical and laboratory facts as encountered by the gastro-enterologist in his daily practice.

It is to be regretted that such a vital function as the digestion seems to rate such a comparatively small percentage of attention in proportion to that accorded the respiratory tract when tuberculosis is present. On the other hand, let me sound a note of warning that the reverse responsibility rests on the gastro-enterologist in that vague gastro-intestinal symptoms not explained by other causes must not be permitted to continue without excluding the possibility of pulmonary tuberculosis.

Cases with gastro-intestinal symptoms as the earlier manifestation of pulmonary tuberculosis are not uncommon and it is to the discredit of the examiner in his original examination if he has not made a thorough examination of his patient to exclude this possibility. It is this type of case, however, that presents the greatest difficulty in diagnosis. The pulmonary lesion is usually early, capable of escaping detection by the stethoscope, or showing only moderate changes in the quality of the breath sounds. There is nothing characteristic in the gastro-intestinal picture. Vague symptoms of gastric discomfort, loss of appetite, a desire to eat but when attempted a feeling of nausea, a revolting feeling toward further ingestion of food, gas possibly present after only a few bites of food are taken, irregular distention, and varying constipation are frequent symptoms encountered. There is no specific reference of the above symptoms to pulmonary tuberculosis; but add to these symptoms exhaustion on rising, a continued sense of fatigue, an unexplainable loss of weight, and secondary anemia, and even though the pulmonary history fails to disclose any positive symptoms we have just cause for growing suspicious of any doubtful pulmonary findings. If further observation of the patient fails to record the expected improvement, the patient should have the advantage of an examination by our consulting Pulmonary Specialist or, where this is not possible, an X-Ray examination. There is scarcely a community so small or so isolated today that recourse can not be had to the X-Ray and, if so desired, a consulting Roentgenologist can be easily consulted by forwarding him the plates if necessary. Too often these cases are permitted to escape the critical eye because the temperature and pulse are normal or, not infrequently, subnormal. One such case presented itself for gastro-intestinal examination requesting that the lungs not be examined because they had been examined within six weeks by the fluoroscope and said to be normal. Our physical examination revealed only very questionably suspicious findings, but X-Ray examination showed early pulmonary tuberculosis. This patient was able to end an invalidism of two years under the care of the Chest Consultant in a very short length of time. Another patient gave a history of gastro-intestinal symptoms for two years with a history of negative chest findings one year prior to our examination, but having moderately advanced bilateral involvement easily recognizable on physical examination at the time of our observation, having unfortunately been treated during the intervening period under the false illusion of nervous indigestion. Another presented the symptoms enumerated above with negative pulmonary history but showed minimal pulmonary findings. This patient when placed under treatment aimed at the

pulmonary condition, with but slight regulation of the gastro-intestinal condition, gained twenty pounds in three months. And still another under observation considered to have a chronic healed pulmonary lesion referred to me by Chest Specialist was allowed to continue under observation for six weeks while showing poor response to treatment, this being attributed to the presence of chronic appendicitis. He was again adjudged inactive on examination by Chest Specialist only to show active lesion on X-Ray.

Be it noted that these patients had normal temperature at four hour checks throughout the day and pulse rate, while active, between sixty and seventy-six.

We may reverse the picture and place somewhat similar symptoms, but add to them marked distention occurring after meals, as the picture frequently seen by the gastro-enterologist in consultation after early pulmonary treatment has been instituted. Is it not strange that in pulmonary tuberculosis, when treatment dictates that the body be put at rest, the mind placed as nearly at rest as possible, that the gastro-intestinal tract is called on to do 100 percent, yea, often times 400 percent more work than it has ever been called on to do before? This, in spite of the fact that its master is under complete rest and that it is absorbing toxins from the existing infection. It is to shudder at some of the sentences imposed on this poor, harmless, unsuspecting organ of digestion when one consults his files to find a history of the previous judge imposing a sentence of two quarts of orange juice and a gallon of milk daily for six weeks, or that of untold cream, or again, that old method of eating all you can and when you can't hold any more, drinking a quart of milk. Is it any wonder that our files are topheavy with cases showing the development of gastric symptoms four to six months after undergoing treatment for their pulmonary lesion? Is it any wonder that the gastric secretory and motor functions frequently affected by inactivity of the patient and toxic absorption revolt under such a regime? Is it any wonder that sensitization built on the background of over-ingestion of particular foods enters the picture to further harass our unsuspecting victim? Is it any wonder that a poor colon forced to undergo such an extra load not infrequently walks the path of spasticity or atonicity?

It is to the credit of the chest specialist of today that such a fundamentally important function as that of digestion is so clearly recognized and appreciated and that in the armamentarium of the modern sanatorium is found the dietician and the consulting gastro-enterologist.

Let us not be so impressed by the usual and more common disorders of low secretory and motor activity as to disregard a most careful history, which may reveal other disturbances in the secretory and motor mechanism, gall bladder infections, allergic manifestations, catarrhal conditions, nervous indigestion, irritable duodenum, or irritable colon. Burning is a symptom that frequently presents itself, and though classically occurring in connection with hyper-acidity, may, when proper analysis of other symptoms are made, be found to be due to depressed secretion probably associated with gastric retention or allergy. Unfortunately it is not always feasible or good judgment to resort to gastric analysis for information and clinical symptoms must be studied carefully.

Drugs for relief of the general condition, toxemia, or to allay the cough are a necessary nuisance to the gastro-enterologist. Anti-pyretics, codein and similar drugs leave their trail of gastric and intestinal disorders. But we must accept them and meet the situation as best indicated.

Cathartics, the bane of every gastro-enterologist's existence, crowd their victims to the front in ever-increasing numbers in the tubercular. Taken from an active life, put at rest, and most likely under forced feeding, constipation frequently ensues. Increasing doses of cathartics with history of having to use stronger and stronger medicines not infrequently results in a spastic or, perhaps more unfortunately, a mucous colitis.

And then we are confronted with the gross pathological lesions of gastro-intestinal tuberculosis. Involvement of the stomach is so rare we may readily ignore it. Involvement is centered chiefly around the cecum, terminal ileum, and colon. Again we are confronted with conditions not manifesting a clear-cut clinical picture. Dr. Emil Granet recently reporting on 2,086 cases, impresses us again with the relatively high percentage of cases, 71 per cent in his series, showing X-Ray manifestations of intestinal tuberculosis with an absence of clinical symptoms. Is this not the answer to the question regarding the difficulty of making a diagnosis in intestinal tuberculosis? is this not a hint to the examiner, granting a given case showing low grade temperature, anorexia, loss of weight not explained by the existing lung condition, to investigate the gastro-intestinal tract even in the face of negative symptoms? And, to give credit to the chest specialist, it may be said that just such investigations have been requested for a considerable length of time.

There is nothing more characteristic than the foul, gray, granular stool containing pus and blood and a diagnosis of tuberculous enteritis is assured, but too late to be of benefit. Through the aid of Brown and Sampson we have for many years been enabled to make our diagnosis at an early date by use of the X-Ray. References to their work on this subject may be readily made. Since the diagnosis depends on visualization of localized spasm, I feel that the fluoroscopic examination should not be relied upon entirely, for in many questionable cases details which can only be observed by plating may be overlooked. The day may not be far distant for the ideal to be realized—a routine gastro-intestinal X-Ray examination for every tuberculous patient regardless of gastro-intestinal symptoms.

Clinical symptoms, unfortunately, are variable. Constipation rather than diarrhea is a frequent early symptom. Vague cramping sensations to sharp pains with variable relationship to meals are frequent symptoms. Gas and distention may or may not be prominent symptoms and in an early case frequently insignificant. Fullness, vague discomfort, and burning over the right lower quadrant are frequently complained of. Nausea, frequently a troublesome symptom, is most often encountered in cases with severe toxemia. Other symptoms less frequently encountered may be elicited from a careful history.

Physical examination may fail to reveal positive findings, but frequently reveals slight to marked generalized tenderness. In tuberculosis of the cecum tenseness over the right lower quadrant may be present. Dull tympany is not infrequently present.

Tuberculous appendicitis and tuberculous peritonitis should always be considered in the differential diagnosis of a given case.

It is not in the scope of this discussion to be able to set forth all of the principles involved in the surgical consideration of pulmonary tuberculosis in its relation to intestinal tuberculosis or of surgery of the tuberculous intestine. Let it suffice to say that since intestinal tuberculosis is so dependent for its progress on the progress of the chest condition that the mere presence of involvement of the intestine is not in itself a contra-indication for chest surgery. Often chest surgery is the means of curing the intestinal lesion. Many factors are to be considered. Careful clinical and X-Ray study of the gastro-intestinal tract should be made before permission for surgical procedure is granted. One other general principle is that operation for appendicitis should hold no fear in itself and should not be too long delayed, but should if possible be absolutely avoided in the face of tuberculous activity of the cecum.

As to treatment—treat the existing gastro-intestinal condition, but in addition, modify this to meet the requirements as indicated by the condition of the patient as a whole. Absolute cooperation between the Chest Attendant and the Gastro-Enterologist is essential. It is my practice to submit my anticipated course of treatment

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Necropsies in Tuberculosis

BY
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PULMONARY tuberculosis constitutes one of the commonest and best known causes of death. With the aid of the X-ray and the microscopic examination of the sputum, the diagnosis may be made with considerable assurance in a very high proportion of all cases of death from this disease. Physicians are therefore often reluctant to request permission for necropsy examinations in instances where advanced tuberculosis has been recognized, and relatives are apt to manifest surprize when it is suggested.

Nevertheless, a post-mortem examination in all instances of death ascribed to tuberculosis should be sought in all seriousness. There are many reasons for emphasizing this procedure. Only too often, afterwards, the physician is reproached for having neglected it. The relatives may well have economic as well as sentimental reasons for wishing the actual cause of death and the complicating factors determined. The physician may have much to learn about his own diagnostic acumen and therapeutic adequacy from the examination of the patients whom he has treated. And patients later to be treated by a physician may well profit from things which he has learned through previous necropsies.

Only too often the fact that a patient has had tuberculosis is assumed as sufficient evidence that this is the cause of death, forgetting the fact that phthisics are just as apt as others to fall victim to homicide, suicide, and accidental death, as well as to most of the other illnesses to which man is heir. Even in the cases where other pulmonary pathology, as coccidioides, cancer of the lung, mycotic infections, etc., may be ruled out, the existence of tuberculosis in the lungs, even in an advanced stage, is not necessarily the cause of death. A study of several hundred necropsies performed at the Olive View Sanatorium, where none but tuberculosis patients are supposed to be admitted, showed that more than ten percent of the patients examined did not have active tuberculosis at the time of death, and another ten percent died of non-tuberculous conditions associated with their pulmonary disease.

Even in instances where the acid-fast bacillus is the cause of the death, the determination of the mechanism or manner in which it acts may be of great importance. Thus, it was found at Olive View that deaths were more apt to occur from complications, intestinal, laryngeal, or pleural, than from the pulmonary lesion itself. The recognition of the importance of such complications is emphasized by necropsy examinations and proves of value to the physician in caring for subsequent patients.

It is not enough, today, merely to know that the lungs are infected by the acid-fast bacillus, but the type of infection, the tendency to exudative or proliferative reactions, the mode of spread and the mechanical factors determining spread and interfering with healing must be considered. The comparison of the clinical and radiographic findings with post-mortem observations is the most important means for improving our knowledge of the pathogenesis and course of the white plague, and is a valuable step in its control.

THE PUBLIC SCHOOLS of our nation have again opened their doors this month. Among the hundreds attending these schools, both city and rural, there are many cases of juvenile tuberculosis. It is generally believed and taught today that most adult cases of the disease result from infection gained during childhood. For this reason the subject of tuberculosis should be taught in each school, as there is no other single subject more important to the school child's present and future health. C.M.H.

The Handling of the Acute Asthmatic Paroxysm

BY
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THE DISTRESS of the patient in an asthmatic paroxysm obviously calls for at least immediate temporary relief. Ordinarily, except possibly in an initial attack, home remedies, such as various inhalants, local chest applications and "smokes," have been tried, and their inefficacy on that particular occasion is the reason for the summoning of medical aid. Such agents as these various nitre smokes, etc., will give relief only in mild attacks, being valueless in paroxysms of any degree of severity, and would hardly be prescribed by a trained physician with decidedly more efficacious remedies available.

The first drug offering enough of a possibility of relief to be worthy of consideration is ephedrine. This is marketed in the form of three-eighths grain and three-fourths grain tablets or capsules of the sulphate or hydrochloride, and as a syrup or elixir containing one-eighth grain to the drachm. It has been the general expression of allergy workers and patients who have used ephedrine, that the three-eighths grain dose will give as much relief as the three-fourths grain, and obviate much of the unpleasant symptomatology frequently accompanying the larger dosage—nervousness, sweating, sleeplessness and, at times, nausea and vomiting. The same symptoms often accompany even the three-eighths grain dose, especially if repeated, and many patients, at least fifty per cent of mine, will not continue with its further use. Except in severe asthmatic paroxysms, its employment subcutaneously is uncalled for, and it is so far inferior to epinephrine in efficacy that its exhibition in such manner is ordinarily superfluous. Given by mouth, it has some value in the milder attacks, especially in children. It is also at times of some help in reducing the epinephrine dosages required to control frequently recurring daily asthmatic attacks. The nervous manifestations of this drug can be considerably abated by combining it with small doses of the common hypnotics.

Also, for many years chronic asthmatics have known of the relief obtainable by the use of proprietary nasal sprays, especially those containing cocaine. The therapeutic action is probably secured by the shrinkage of the vasomotor rhinitis mucosa so often accompanying the asthmatic paroxysm, and the deadening to some extent of the reception of nasal sensory stimuli that would incite the reflex bronchial spasm. While practically there is very little possibility of cocaine addiction to be invited by such usage, such possibility is at least potential. Also, a certain percentage of asthmatics are very sensitive to cocaine, novocaine and allied products, and unpleasant reactions can occur. Often identical helpful results can be secured by the employment of a one per cent. ephedrine nasal spray, and while sensitivity to ephedrine is not unknown, only very few cases have been reported, and its use as a nose spray is certainly very gratifying in stopping mild asthma or asthmatoid bronchitis, especially in children, and in my practice is almost routine until proper permanent measures can be instituted for the control of nasal turgescence.

Atropine, belladonna, iodides, calcium and glucose given by mouth, hypodermatically or intravenously, while of some value in isolated cases, have, in the hands of most allergy workers, proven, on the whole, inefficacious.

This brings us up now to epinephrine, undoubtedly the most reliable remedy for immediate asthma relief yet discovered, and a real boon to suffering allergic humanity. Given hypodermatically in proper dosage (the average dose is 0.5 c.c.), relief comes within a few moments, and if not complete, usually becomes so with

a repetition of such dosage at the end of twenty minutes to one-half hour. Fortunately, this remedy so employed, and repeated if necessary in this manner every few hours, will, in the vast majority of instances, give the desired relief. The case is then ready for the ordinary standard methods of etiological diagnosis and treatment to prevent recurrences.

Now, unfortunately there exists a percentage of asthmatics in whom, on account of unusual attack severity, no such relief whatsoever is secured by repeated epinephrine doses or in whom such relief is partial and transient, and such frequent repetitions of the drug in large dosages are required that its further use becomes either intolerable or unavailing. Under these, and these circumstances only, is our next standby, morphia, necessary or justifiable, not only from the possibility of inviting addiction, but also because many asthmatics are actually and decidedly sensitive to any or all opium derivatives, showing this sensitivity in subsequent intense nausea and vomiting, and following temporary relief, recurrence of the asthma. For this reason, at least fifty per cent of my patients rebel against its use, and will accept it only as a last resource. However, under the conditions mentioned, occasionally its use is absolutely necessary.

Now let us presume that both epinephrine and morphia in frequent and rather massive dosages have given no, or only partial temporary, relief, and that the patient is in a constant or almost constant severe status asthmaticus, which, if not broken by some means or other, may in a few hours or in a day or two terminate in death from exhaustion. There is scarcely in the catalog of human suffering any picture more pitiful than one of these sick asthma cases in this condition, and scarcely one in any other ailment in a more critical state, as death is far from unknown in these patients. It must be remembered that at this stage some of these cases are unconscious and almost pulseless, and the unconsciousness is not by any means invariably due to morphine overdosage, though, of course, this must be taken into consideration. The amount of pupillary contraction is a guide.

For such desperate conditions, four surgical methods have been suggested:

1. Cervical sympathectomy. I have seen one case, a failure, afterwards relieved with no great difficulty by usual conservative asthma treatment. This operation has been discarded.

2. Alcoholic blockage or section of the dorsal sympathetics. This operation has given temporary relief, but is scarcely yet to be accepted as safe or a standard measure on account of the limited number of cases reported in the literature.

3. Bronchoscopy. A number of cases have been temporarily relieved by this line of treatment.

4. Immediate surgical operative work on paranasal sinuses. Which cases of asthma will require ultimate surgical handling of the paranasal sinuses to secure permanent relief is not the purpose of this article. The cases I am discussing are in such condition that any surgical interference for the time being, except possibly dorsal sympathectomy, would not be tolerated.

As far as I can express an opinion, all these measures are superfluous as it is probably over five years since I have been unable to give relief to this condition with a case of this type to handle every few weeks.

Also, the not infrequent, at least temporary, abeyance of asthma attacks during pregnancy, some non-related surgical operation or an intercurrent non-respiratory tract infection accompanied by a period of high bodily temperature elevation, has led to efforts to secure such relief by the production of high temperatures artificially. This has been done most often either by subcutaneous or intravenous injections of peptone, milk, typhoid vaccine, and more recently by diathermy. Results have been inconsistent and unsatisfactory.

What then can be done?

In order at this state to secure any results whatsoever, there must exist a little knowledge and consideration of the physio-pathological processes at work. There

are three possibilities. First, there has been such a massive and continuous overdosage of specific and non-specific antigenic stimuli that the ordinary defensive immunological mechanisms of the individual involved against such stimuli have been completely overwhelmed for the time being. Or secondly, the sympathetic nervous system controlling bronchial spasm is in such an exquisite state of hypersensitivity that minor stimuli that would probably be of no effect, maintain the spasm with a vigor that our ordinary antispasmodic agencies will not control. Possibly both factors are at work, but fortunately can be handled by identical measures. Thirdly, there is always the additional possibility of spasm maintenance by the physical efforts due to ineffective coughing efforts to expel heavy, viscid mucus, and casts, which actually threatened to induce partial atalectasis and suffocation, a condition not rarely seen post mortem in deaths from bronchial asthma. In my hands so far, handling the first two possibilities has ultimately taken care of this contingency.

Naturally the handling of such a case calls for the elimination of practically all known specific or non-specific antigens or stimuli that will induce or maintain the asthma paroxysm. A private hospital room (not a ward bed) maintained at an even comfortable temperature is almost essential. Such a room is then made, and meticulously kept, antigen free. Pollen and ordinary outside atmospheric dusts are taken care of by suitable filters or excluded by closed and tightly sealed windows. House dusts, such as those derived from feathers, wool lint or fuzz, animal hair, furs, cosmetics, insecticides, must be taken care of, and also smoke and odors of all kinds.

Often only by the perfect or near perfect elimination of such stimuli is there the slightest possibility of recovery. The slightest physical exertion in these cases is usually impossible without producing or increasing the asthma, and its trial must be absolutely forbidden. Fortunately, there are no dietary worries at this time as these cases cannot take nourishment or even water. Special nursing care is absolutely essential.

Once this environment has been accomplished, a return to epinephrine and morphia, given frequently under careful supervision, will usually after several hours of within a day or two now break the asthmatic status, where they had previously proven unavailing. However, even under such ideal conditions, not a particle relief may come. In such a desperate situation with wild spasms persisting, the patient only partially conscious, pale, trembling and almost pulseless, with a terminal exhaustion about to supervene, and pin point pupils forbidding further morphia, the proposition seems, but rarely is, hopeless. Fortunately, there are other measures available, one of which, if repeated sufficiently frequently will in the end break the spasm.

First comes epinephrine given slowly intravenously in from two to four minim doses every ten, twenty minutes or half hour. The employment of the 1:10,000 dilution in this connection is as efficacious as the use of the usual stronger 1:1,000 dilution, and avoids the decidedly uncomfortable epinephrine symptoms of headache, tremor, pallor and vomiting. Relief usually ensues. It is remarkable how promptly and completely such small intravenous doses give relief after massive 1 c.c. doses every fifteen minutes or half hour subcutaneously have completely failed. There exists the possibility that with the retarded circulation seen in these cases with weak, thready pulse, the subcutaneously administered epinephrine is absorbed from the tissues so slowly that partial or complete digestion or chemical alteration occurs, causing the loss of most or all of the antispasmodic radicals.

Direct inhalation anaesthesia by ether, chloroform and carbon dioxide for this status asthmaticus has been reported as giving some results, but literature reports and my personal experience are too meager to pass any opinion. If any general anaesthetic is to be tried in these cases to relax spasm, a warning should be given regard-

ing the choice of sodium amytal to be used intravenously. Theoretically under these circumstances, the drug would seem indicated, but two deaths have been reported from its use in asthma, and I have had one case decidedly too close to an immediate fatality to be comfortable. Anaesthesia produced by ether and olive oil given by rectum, as originated by Maytum of the Mayo Clinic (1), has worked gorgeously for me in some instances and failed dismally in others. This procedure can be repeated in a few hours if necessary, and can be used coincidentally with the intravenous epinephrine with happiest outcome.

When the extreme status asthmaticus has been broken in these patients, one is not through with them by any means. Even under the most meticulous observation and elimination of all known asthma antigenic stimuli, the usual story of these cases even in antigen free rooms is a gradual reduction of the relief measures employed. Epinephrine in reduced doses may be required for several days, gradually dropping to two or three doses a day from the eighteen or twenty doses required at the inception of treatment, and a single nocturnal dose may be required for weeks. For some time these cases may be in such a state of extreme hypersensitivity that the slightest infraction of antigenic precautions results in severe relapses.

When these cases are able to take nourishment, the dietary precautions should consist in the avoidance of all foods known to have antigenic properties, lists of which are easily available. Drugs also fit into this category.

Many of these cases will now show a blood eosinophilia previously absent.

A word of warning should be given regarding testing these sick cases with our usual methods. These tests should not be attempted until these patients are practically ambulant convalescents at least and epinephrine reduced to a minimum. If we regard the positive skin reaction as indicative of some measure of immunological response, as we regard the positive tuberculin test in tuberculosis, then precisely as happens with the employment of the latter agent in advanced tuberculosis, the tests are not only practically invariably negative, but each represents a potential antiguen dosage that might induce a severe protracted relapse. This is especially true if pollen material is used. This statement is made as a result of some bitter personnal experiences after too early or too strong testing. Inasmuch as all known causes of asthma are being taken care of in these cases, there exists no necessity for immediate testing as far as immediate relief is concerned. Often the history of a case will admit the possibility of accurate enough diagnoses to admit specific treatment inception before any positive tests can be secured, and often this will be necessary, as weeks or months may be required before tests will become positive in these cases. Such initial specific treatment naturally must start with an almost infinitesimal dilution and be proceeded with very cautiously as there will be no local arm reactions to warn of the approach of such overdosaage and subsequent relapse.

Living under a total, or nearly total, antigen free environment will ultimately clear these chronic severe cases, it might be said, practically without exception, though, unfortunately many weeks or months, or even in extreme instances a year or longer, may be required. But the results are worth the time invested, and if tuberculosis cases can give months to sanatorium residence to regain health, there is no reason why a similar period of time cannot be given by a severe chronic asthmatic invalid. Ultimately by persistent intelligent care and appropriate desensitization methods, a high degree of permanent relief and economic recovery should be secured with this severe type as well as the usual milder types of bronchial asthma.

REFERENCE

(1) Maytum, C. K.: Bronchial Asthma: Relief of Prolonged Attack by Colonic Administration of Ether, Med. Clin. North America, 15, 201, July, 1931.

Treatment of Intestinal Tuberculosis With Especial Reference to Oxyperitoneum

BY
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DURING THE PAST two decades our conception of the etiology, diagnosis and treatment of intestinal tuberculosis has changed considerably. Our present knowledge indicates that, for practical purposes, etiologically, it is secondary to active pulmonary tuberculosis of the ulcerative type. Illustrative of this opinion, Boles and his associates in 226 autopsies of patients with tuberculosis of the lungs concluded that where there were no pulmonary ulcerations there were no intestinal ulcerations. We must keep in mind, however, that the early lesions of intestinal involvement are not ulcerations, but microscopic tubercles. It should also be remembered that intestinal tuberculosis does occur fairly frequently as a complication of early pulmonary tuberculosis.

Perhaps the greatest handicap we have at the present time in regard to intestinal tuberculosis is our lack of a definite, practical means of diagnosis. We know that in at least a third of the cases with intestinal tuberculosis, even where the lesions are very extensive, there are no symptoms or signs which draw our attention to the abdomen. Again, we know that when symptoms or signs do occur, they are not proportional to the extent or degree of involvement. The early lesions may give severe symptoms and the symptoms of advanced lesions may be mild or absent.

It is also understood at the present time that tuberculosis of the other abdominal viscera, especially the spleen, liver, kidneys, peritoneum and suprarenals, does quite frequently exist as a complication of active pulmonary tuberculosis in conjunction with or in the absence of intestinal involvement. Physical signs and symptoms, then, as a means of diagnosis of this condition have been found wanting.

About eighteen years ago the X-ray study of the intestinal tract, following the barium meal, was introduced as a means of diagnosing intestinal tuberculosis. For a time it was felt that our problem was largely solved. Experience has proven otherwise. Further studies, particularly those based upon necropsies, reveal that a large percent of our intestinal cases still go undiscovered.

More recently, Boles and Cohen have given enthusiastic reports of the value of the double contrast enema in the diagnosis of tuberculosis of the bowels. We have yet to learn the value of this method.

From a clinical point of view, perhaps the most important thing that we have learned about tuberculosis of the intestine is that it is a curable condition; that it is curable whether it exists as a complication of early or of advanced pulmonary tuberculosis. The exception to this statement is where tuberculosis of the bowel is part of a general hematogenous infection or where it exists in conjunction with tuberculosis disseminated throughout the other abdominal viscera.

Evidence of a healed tuberculous ulcer of the bowel has, in the past, been regarded as very rare. Gardner, as well as others, has seen them frequently. I believe that those of us engaged in the handling of large numbers of tuberculous patients have many times seen tuberculosis of the bowel, demonstrated by laparotomy, existing as a complication of extensive pulmonary tuberculosis, become clinically well and remain well.

As has been suggested, the treatment of intestinal tuberculosis is not as satisfactorily understood as it perhaps would be if we were able to be more definite in our diagnosis of this condition. There is more or less of a general feeling at the present time that a certain percent of the cases complicating early pulmonary tuberculosis get well as a result of the general treatment of the pulmonary condition. It is also felt that even some of the more extensive

cases occurring with early or with fairly extensive pulmonary involvement similarly get well.

The usual special routine of protection of the abdomen (keeping it warm), non-irritating diet, etc., does relieve a certain proportion of these patients symptomatically, but they, in themselves, are not very important as curative measures.

Heliotherapy, both natural and artificial, as originally outlined by Rollier and modified for use in cases complicated with active pulmonary tuberculosis, used extensively in this country and abroad, has, where it has been possible to carry it out scientifically, been credited with symptomatic relief as well as cures in a large percentage of the cases treated. The one drawback to this method of treatment is that there are a lot of patients who are either too sick with their pulmonary involvement or are so situated that it is impossible to scientifically apply heliotherapy in any form.

Surgery in the treatment of intestinal tuberculosis has, more or less recently, had its day, but has been found wanting. The important drawbacks in this method of treatment were the frequency of extensive bowel involvement, the involvement of other abdominal viscera and the inability of these patients to withstand the resulting serious shock of major surgery.

The introduction of oxygen into the abdomen, or oxyperitoneum, in the treatment of tuberculosis of the abdomen is not new. It was used in Germany, France, England and India in the early part of the century and has been used fairly extensively in this country during the past twenty-five years, being originally popularized by Bainbridge and Meeker of New York.

The reports of the men who have intelligently and persistently applied this method of treatment of intestinal tuberculosis have always been very encouraging both as to symptomatic relief and as to cure. Recently Banyai of Wauwatosa Sanatorium in Milwaukee has reported in detail some fifty cases of intestinal tuberculosis treated by oxyperitoneum. These were all cases unrelieved by other methods of treatment. There was a satisfactory result with this special treatment in 85% of those treated.

I have used this method of treating intestinal tuberculosis for the past twelve years. It has been used on my services in the Orange County Sanatorium and the Los Angeles General Hospital as well as in private practice. It has not been used to the exclusion of other methods of treatment, but I feel it is, in general, by far the most satisfactory adjunct in the handling of these conditions.

The factors which recommend it are, first, the simplicity of application. It can be carried out either in the office or at the bedside with the ease of a pneumothorax refill. Second, it can be used no matter under what circumstances or in what environment the patient may be compelled to take the cure. Third, it can be used as a diagnostic measure. As has already been mentioned, the diagnosis of intestinal tuberculosis, even under the best circumstances, is fraught with a good deal of uncertainty. Experience warrants the feeling that if patients suspected of intestinal tuberculosis are relieved by oxyperitoneum, our suspicion is justified, and vice versa. There are no particular preparations for or contraindications to its use. With proper technique, if the patient is not benefited, he is not harmed by this treatment.

The technique, which has previously been described, is very simple. In twelve years' use in hospital and private practice I have never experienced any bad results.

I am reporting a few cases in detail which illustrate, in general, the type of case treated and the results obtained.

Mrs. S., aged 28, advanced, bilateral, fibro-caseous pulmonary tuberculosis. Severe abdominal symptoms, simulating subacute appendicitis. Laparotomy disclosed extensive tuberculosis of the appendix with intestinal involvement extending both sides of cecum. Operation followed by oxyperitoneum as follows:

May 16,'29	250cc.	Aug. 3	400cc.
May 30	400cc.	Sept. 4	370cc.
June 14	400cc.	Oct. 13	400cc.
July 8	400cc.	Mar. 27,'30	400cc.

No other treatment for intestinal condition. After the first two or three treatments patient always asked for the oxyperitoneum when she felt the need of it. Patient well and leading a normal life for the past five years.

Miss M. DeP., aged 27, advanced, bilateral, fibro-caseous pulmonary tuberculosis. Abdominal symptoms simulating fairly acute appendicitis. Surgery revealed extensive tuberculous involvement of the appendix with considerable involvement of the bowel on each side. Appendix was removed and oxyperitoneum given fifteen times in amounts varying from 300cc. to 600cc. over a period of a year and a half. No other treatment for the abdomen. After the first two or three injections, patient asked for the treatment when she felt the need of it. Patient well and living a normal life in North Dakota the past seven years.

Miss M. C., age 32, chronic, fibro-caseous pulmonary tuberculosis with fibrosis predominating. Abdomen opened. Tuberculous appendix removed. Some tuberculosis of the bowel limited more or less to the cecal area. Oxyperitoneum induced and given twenty-three times in amounts varying from 350cc. to 1200cc. over a period of two years. No other treatment. Prolonged treatment is due to patient's asking for the injection. Patient well for the past five years.

Mr. F. F., age 26, advanced, bilateral, fibro-caseous pulmonary tuberculosis, caseation predominating. Severe abdominal distress, diarrhea (10 to 15 bowel movements a day). Oxyperitoneum given as follows:

Apr.9,'25	300cc.	Apr.23	400cc.
Apr.17	350cc.	May 1	500cc.

No other special treatment except rest. Abdominal distress relieved; bowel movements reduced to one or two a day, more or less normal in character. Pulmonary condition progressed; abdomen remained comfortable.

Mrs. C. E. S., age 30, progressive, advanced, bilateral pulmonary tuberculosis. Came from outstanding sanatorium. History of 17 bowel movements a day for two months, loss of 24 pounds in that time, all treatment for intestinal condition ineffective. Oxyperitoneum given eleven times in amounts varying from 300cc. to 400cc. over a period of eleven months. No other special treatment given except rest. Bowel movements reduced to one or two a day after the first injection, and abdominal distress relieved although pulmonary condition progressed to death.

Mrs. E. E., age 40, acute, exudative, unilateral pulmonary tuberculosis, rapidly progressive. Pneumothorax controlled lesion in the lung. Acute abdominal distress developed; 8 to 15 bowel movements a day. Oxyperitoneum was given as follows:

Feb.22,'34	300cc.	Mar.3	350cc.
Feb.26	350cc.	Mar.15	350cc.

No other treatment except rest. Intestinal condition was promptly relieved and patient went on to recovery without further disturbance.

Miss N. W., age 18, acute, unilateral, exudative, advanced pulmonary tuberculosis. Acute abdominal distress with diarrhea developed as part of a general hematogenous infection. In spite of a well established pneumothorax, the disease went on to a fatal termination. Oxyperitoneum was given twenty-five times about a week apart, in amounts varying from 300cc. to 500cc., over a period of nine months. There was marked relief of the abdominal condition in the early part of the treatment. Toward the end, due to systemic involvement, there was very little relief secured other than through the psychic effect.

The Mediastinum

IN CONSIDERING the pneumodynamics of collapse therapy, the mediastinum must come in for a major share of attention. We must visualize the thorax as three-chambered with the central chamber, or mediastinum, walled off from the pleural cavities by two elastic partitions.

AN OPERATION FOR TUBERCULOUS EMPYEMA . . . . (Continued from page 9)

a little air from the thorax and causes a gradually increasing negative pressure in the empyema cavity, for it is more difficult for air to enter through this valve-like wound than to escape from it. This fact is readily demonstrable (although in theory the valve might seem to work the other way) if the wound is opened by an instrument at a change of dressings, when air will enter it with a sucking sound. If much negative pressure is desired, over-zealous and overcleanly nurses should be cautioned not to change the dressings or to content themselves with changing the outer ones only and to leave the inner ones in place as long as possible—for many days.

This operation, it will be seen, differs entirely both in theory and practice from the valve recently proposed by Nichol for acute empyema. Details, with X-rays, case histories, and other data will soon be published.[1]

[1]Western J Surg.

THE CAUSE OF TUBERCULOSIS . . . . (Continued from page 11)

tubercle bacilli taken into the body at any one time, but is to a great degree determined by the physical condition of the individual. For example, a person who is in a run-down or debilitated condition because of over-work, poor food, unhygienic living conditions, worry, or by having recently had some other disease, such as influenza, is sure to have a much more extensive reaction than a person who is in the best of health. It has been shown repeatedly that tuberculosis and poverty, with its resulting poor living conditions, go together: the poorer the living conditions, the greater the incidence of tuberculosis. The great destructiveness of tuberculosis among primitive races, such as Mexicans, Negroes, and Indians, has now been shown to be caused largely by their unhygienic and inadequate living conditions, and not due to racial susceptibility. That is, people of this type generally live in small, overcrowded houses which are poorly ventilated and where the food is of the poorest quality. In short, they live under conditions which markedly lower their resistance to disease and which at the same time favor the spread of germs. Environment is, then, a most potent force in shaping the course of tuberculous infection, just as it is in presenting opportunities for the reception of infection.

SUMMARY

1. Tuberculosis is caused by tubercle bacilli (germs) which are transferred from a consumptive to a well person, usually in sputum.

2. Tuberculosis is not hereditary; i. e., a person is not born with the disease.

3. Tuberculosis runs in families merely because the chances of acquiring the germs are so much greater under such intimate relationships.

4. Tuberculosis is not acquired solely in childhood; no age is exempt.

5. A child or an adult usually overcomes the first infection with the production of a partial immunity to subsequent infections.

6. The immunity developed by a first infection may be broken down by massive doses of tubercle bacilli or by adverse living conditions.

7. It is the reinfection which takes place after the immunity of a first infection is broken down that produces active tuberculosis.

8. Environment is the most important force in promoting infection and in shaping the course of an infection with tubercle bacilli.

to the Chest Specialist before instituting it, so there will be no conflict in treatment. I am accorded the same courtesy and co-operation in my referred cases.

A diet of high caloric value is immediately instituted for emaciated and underweight patients. Unfortunately this type of diet is frequently taken to apply to all tuberculous patients, with the result that we see a patient of normal weight growing obese and the occasional obese patient further depressing his resistance by growing more obese. A patient put to bed, inactive, and gradually losing his toxicity frequently develops an appetite which must be guarded against if excessive weight is to be avoided. It is not uncommon to see a patient with a minimal healed lesion showing evident shortness of breath resulting from excessive weight. Furthermore, gastric symptoms follow in the wake of ill-advised stuffing. Increased caloric value may readily be obtained with intermediate feedings reinforced with the many carbohydrate preparations available. The patient with a continued poor appetite is a problem to all. Our complete list of bitters, appetizers, and so on, are often of no avail and we find ourselves still floundering in our attempts to produce artificially what nature has decreed against. I have used insulin with only indifferent success, but continue to use it if apparently indicated. In tuberculous enteritis a smooth high caloric diet fashioned after that of Alvarez, or in the more severe cases a diet consisting of gruels, purees, and the more highly nourishing liquids, and soft foods is advised. A diet rich in vitamines is now in use. The prepared commercial combined vitamine products, or tomato juice and cod liver oil as recommended by McConkey may be used as additions to the diet. In patients showing allergic tendencies the diet should be arranged to meet the requirements necessary; however, a loss of weight from the normal can not be justified therefore precluding the rigid diets one might desire to prescribe.

Light therapy properly administered is of advantage.

Medicinal agents are to be prescribed as indicated. When intestinal lesions are present one of the several kaolin or aluminum hydroxide preparations available are of benefit. Anti-spasmodics and sedatives are to be prescribed as indicated. Calcium in any of the assimilable forms is readily and frequently prescribed in my regime of treatment.

In conclusion, let me emphasize:—

1. The importance of considering chest pathology in gastro-intestinal cases presenting vague symptoms not responding as one would expect, especially if we elicit a history of unexplained loss of weight, undue fatigue, hypotension, low grade temperature, or anemia.

2. That gross tuberculous intestinal lesions may be so insidious in their onset that unless this possibility be kept in mind serious consequences may result.

3. That, as goes the chest, so goes the intestinal tract.

4. That, in reasonable bounds, intestinal involvement is not a contra-indication to chest surgery.

5. That non-tuberculous conditions of the gastro-intestinal tract are frequently precipitated by over-zealousness in forced feeding.

ABSTRACTS

This department is devoted to abstracts of articles carefully and judiciously selected by the Editorial Staff.

BREUER, MILES J.: Tuberculin Therapy. Annals of Internal Medicine. Vol. 5, No. 7, p. 1447.

For a number of years there has been considerable controversy over the efficacy of protein extracts of tubercle bacilli in the treatment of tuberculous patients. Successful results are ardently claimed by some, and denied with equal emphasis by others. The author believes that we have at present a sufficiently comprehensive conception of tuberculous allergy and tuberculous immunity to enable us to formulate at least a tentative working basis for the scientific use of tuberculin therapy.

Immunity is that characteristic of the tissues by virtue of which tubercle bacilli are not able to survive there. A subject with good immunity survives infection with tubercle bacilli, whereas one with low immunity dies quickly from the infection. Allergy is a sensitization of the subject's tissues to initial inoculation of tuberculoprotein by virtue of which subsequent inoculations produce inflammation in these tissues. The nature and severity of the symptoms will depend on the degree of sensitization that has been established, and the size of the subsequent doses. A subject rendered allergic by initial inoculation is also rendered to a greater or less degree immune. Yet, immunity and allergy are not the same thing; they do not even run parallel, but are totally independent of each other.

Injection of tuberculoprotein (tuberculin) has only a negligible effect on immunity. Tuberculin, therefore, cannot be expected to produce a cure by increasing the immunity of a subject. The effect of the injection of tuberculin into the animal body is to produce the allergic state. Subsequently, the injection of a large dose will produce a severe reaction, with inflammation, exudation and necrosis. But, a series of smaller doses, properly graduated and timed will de-sentitize the individual and diminish the allergic state, so that larger and larger amounts of tuberculoprotein can then be administered without systemic or local disturbance. Therefore, in the treatment of a patient, tuberculin can only be used to reduce the allergic sensitization.

Allergy, however, is at the bottom of a large proportion of the patient's illness and clinical symptoms, of his distress and discomfort. His fever, his malaise and reduction in capacity for effort, his lack of appetite and loss of weight, are all allergic phenomena. Allergy is responsible for the inflammatory process with its exudation and its necrosis. The whole "toxic" and inflammatory picture is due to allergy. It is completely absent in non-allergic animals dying from lethal doses of tubercle bacilli.

The author reports 26 cases selected for their allergic symptoms—treated with tuberculin. These cases were controlled in that they had previously failed to respond to general therapeutic regimen. Seventy-six percent of these subjects showed improvement under tuberculin. As a contrast there is also reported 181 uncontrolled, allergically unselected cases in which only 43.6% showed improvement under tuberculin.

The effect of tuberculin is to decrease the patient's allergic sensitization. Clinically it ought to be useful in those cases where the allergic state is principally at the basis of the patient's symptomatology.

DAVIES, DANIEL T., HODGSON, H. GRAHAM AND WHITBY, LIONEL E. H.: A Study of Pneumococcal Pneumonia. Lancet, 228: 919-924 (April 20) 1935.

This paper is based on the radiologic examination of 119 cases of pneumococ-

cal pneumonia, and the results have been correlated with the clinical and bacteriologic findings; during the investigation approximately 1,500 radiograms were taken.

Type I. Pneumococcal Pneumonia.—Type I pneumonia is the classic variety: It is sudden in onset and the physical signs during the illness are usually well defined. It is not surprising therefore that the x-ray appearances are also distinct and uniform. Of the Type I cases, 37 were followed through the infection.

It will be convenient to consider separately those which had no specific treatment (18) and those which had Felton's serum (19).

Untreated with Serum.—Early signs: Of this group, two were radiographed within 30 hours of the onset, and in both of them x-rays showed consolidation on both sides of the chest, whereas clinical examination had suggested that only one lung was involved.

The established consolidation: The opacity spreads outward from the hilum, involving the periphery last, and varies in density from hazy to dense. The authors have used the term "hazy" when the line of the diaphragm could be seen and "dense" when it could not. Of the 18 patients, only four gave hazy shadows; in two of these no physical signs could be detected and in the other two they were not characteristic of consolidation. In the 14 in whom the opacity was dense, the x-ray and clinical findings corresponded closely. In four of these, however, radiography showed more extensive disease than was indicated by clinical signs. It was somewhat surprising to find that in only eight out of the 18 was the pneumonia confined to one lobe, although clinically there were only two with definite signs that more than one lobe was involved. Consolidation was maximal on the seventh to eighth day. The average day of crisis was the eighth.

Resolution: As soon as resolution begins—and not before—the hilar glands on both the affected and unaffected sides show obvious enlargement. Accordingly, when a case is radiographed some 10 days after the onset, it is possible to give an opinion whether resolution has or has not begun. The glandular enlargement which takes place during resolution is quite unlike the enlargement due to old pulmonary disease, where the glands are irregular in outline; here they are globular and appear swollen. The time required for complete resolution is very variable. Some lobes have appeared normal 10 to 14 days after the onset, while others have shown a delay up to the fiftieth. The average for the untreated Type I group was 20 days from the beginning of the illness. The rate of resolution presumably depends on the extent of the consolidation and its density. The authors' experience is that enlarged hilar glands are associated with rapid resolution, whereas calcified glands—the result of past disease—are usually associated with some delay.

Complications: Two patients who developed empyema showed dense consolidation from the outset. Clinical signs were more reliable than the x-ray appearances in revealing this complication. It would be irrational to expect radiography to disclose a small layer of pus in an area already opaque through consolidation.

Severity of illness: Although there were exceptions, the authors came to regard a dense consolidation as being usually associated with severe constitutional symptoms; a hazy opacity on the other hand was characteristic of the milder forms of pneumonia. Of 18 cases, eight were classified as mild, and of these, four showed hazy opacities. The milder the case the more rapid the resolution.

Extension of consolidation: Only one case in the Type I group showed an extension of the pneumonic process. Despite an early crisis on the fourth day the initial consolidation in the right lower lobe was slow in clearing, and there was much enlargement of hilar glands. From the fifteenth day onward there was a gradual extension of the left hilar shadow which ultimately involved the whole of the left upper lobe. This gave typical physical signs four days after it was first

noted on the x-ray films while the initial consolidation was resolving this shadow extended, and it remained persistent for some considerable time. The patient was radiographed as late as the 170th day, and even then some shadowing was still present. While a rapidly forming consolidation often as rapidly disappears, a slowly forming one frequently ends in delayed resolution and sometimes permanent fibrosis.

Serum-Treated Cases.—Early signs: Of the 19 in this group, five were radiographed within 48 hours of the onset. In all five a definite opacity was seen spreading from the hilum to a lower lobe. The physical signs were equally clear and no difficulty was experienced clinically in outlining the lobe involved, although in one case a smaller area of consolidation on the opposite side was only detected radiologically at that time.

Established consolidation: Dense opacities were observed in 14 of the cases at the height of the illness, while the other five showed only a hazy consolidation and atypical physical signs. In five cases the clinical signs did not indicate the full extent of the lung involvement, and it is important to note that in four of these it was an upper lobe consolidation that was missed. The physical signs of upper lobe pneumonia are, in fact, surprisingly few.

The effect of serum treatment is to shorten the febrile period and induce an early crisis, but no material hastening of resolution was observed in these cases. Indeed, some with few signs of consolidation when serum treatment started on the first day showed progressive consolidation despite an early crisis on the third day. The average day of crisis in the first 10 cases which were treated in the second and third day of their illness was the fourth to fifth, whereas the clinical and x-ray signs reached their maximum on the seventh. Although the number of cases is small, the authors feel justified in stating that, in spite of the dramatic relief afforded by serotherapy, it has no demonstrable local effect on the consolidation.

Resolution: The most rapid resolution was in a patient treated with serum on the second day. The crisis was on the third, and no radiologic signs could be seen on the seventh. In four which showed long delayed resolution—up to the ninetieth, fifty-ninth, fifty-first and thirtieth days—calcification of the hilar glands was present. Of these patients, nine suffered from chronic chest disease, and the average day of complete radiologic resolution among them was the thirty-first from the onset of the illness. In contrast, seven patients who were previously well and who were free of complications showed complete radiologic resolution, on the average, by the fourteenth.

Complications: There were three cases of empyema in this group, and the one which was fatal showed bilateral empyema. In all three the consolidation was very dense from the outset.

Extension of consolidation was seen in only one case. The process eventually involved all of both lungs except the right upper lobe.

Type II. Pneumococcal Pneumonia.—Type II pneumonia is more severe than Type I. The toxic pallor and the quiet resignation of the patient contrast forcibly with the high color and restlessness of Type I infection. The onset is sudden and the signs of consolidation are usually well-defined.

Seven patients were studied from the fourth day onward. The general x-ray appearances differed in no way from those seen in Type I, but whereas in the normally progressing Type I infection the lung cleared up completely, leaving no trace of the inflammation, in Type II, there seemed to be a tendency to a permanent, although slight, increase in the density and extent of the pulmonary striae.

The relation between clinical and radiologic signs was more or less close. The infection was confined to one lobe in four of the patients; in the remaining three (two of whom died) two lobes were consolidated. Resolution was complete in a period varying from 12 to 31 days. Enlargement of hilar glands was observed as in Type I. (Continued in November issue)

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CASE REPORTS*

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LEUKOPENIA IN TUBERCULOSIS

By C. H. C.

A young man, aged 26, weight 132 pounds, height 5 feet 10 inches, was first seen in October, 1927, with extensive bilateral cavernous tuberculosis, worse on the left. His hemoglobin was 85 per cent; red blood cells were 4,410,000; white cells, 8,375, with neutrophils 70 per cent.

Left-sided artificial pneumothorax was necessitated by repeated hemorrhages. In June, 1931, clear serous fluid developed. During its removal with an 18-gauge needle July 3, 1931, the patient contracted his muscles, breaking the needle at the hilt. It was removed under fluoroscopic control at the Biltmore Hospital. The maximum temperature for three days was 100° F. There was no wound infection.

July 11, the patient awoke with a temperature of 102.2° F., headache, prostration, nausea, and severe abdominal pain. No relief was obtained by ice bag or enemas. There was no rigidity, no tenderness, no jaundice, no enlargement of the lymph nodes. The spleen was not palpable. July 13, the leukocytes were 4,300, with 90 per cent small lymphocytes and no polynuclears. The pharynx was congested, but he complained of no pain or discomfort. Pentose neuclotides were not available; no response was obtained from leukocytic extract. July 16, the leukocytes had fallen to 1,200; no granulocytes were present. The throat was sore; only congestion was visible. He returned to the Biltmore Hospital, and his temperature ranged from 102 to 104° F. July 17, 500 c. c. of whole blood were transfused. His hemoglobin was 80 per cent; red blood cells were 4,190,000; white blood cells, 1,600; sm. lymphocytes, 85 per cent, with no polynuclears. The throat was sorer, but showed no ulceration. July 21, white blood cells were 1,450 with no polynuclears. Roentgen radiation was administered over long bones, after which the leukocyte count was 2,400, with 4 per cent polynuclears, this being the first time in eight days that the granulocytes had been seen in the peripheral blood stream. Radiation was repeated two days later. The leukocytes rose to 6,350 with 50 per cent polynuclears, and there was marked symptomatic improvement. July 25, an ischiorectal abscess was discovered. The white count was normal. The abscess was evacuated; convalescence was normal.

Without remission he continued improvement in his general and tuberculous condition. He returned to his home in Indiana in July, 1932, one year after this episode and has remained well since.

The case is interesting because of the length of time that granulocytes were absent from the peripheral blood stream, longer than is considered compatible with life. The only possible etiological factor was continued self-administration of mild doses of allonal, unknown to me.